ARIZONA STATE VETERINARY MEDICAL EXAMINING BOARD
1740 W. ADAMS ST., SUITE 4600, PHOENIX, ARIZONA 85007
PHONE (602) 364-1PET (1738) FAX (602) 364-1039
VETBOARD.AZ.GOV

COMPLAINT INVESTIGATION FORM

If there is an issue with more than one veterinarian please file a separate Complaint Investigation Form for each veterinarian

PLEASE PRINT OR TYPE

FOR OFFICE USE ONLY

Date Received: Sept. 17, 2020 Case Number: 21-29

A. THIS COMPLAINT IS FILED AGAINST THE FOLLOWING:

Name of Veterinarian/CVT: Dr. Stephanie Foote

Premise Name: VetMed Arizona

Premise Address: 20610 N Cave Creek Rd.

City: Phoenix State: Arizona Zip Code: 85024

Telephone: 602-697-4694

B. INFORMATION REGARDING THE INDIVIDUAL FILING COMPLAINT*:

Name: David Evers

Address: [redacted]

City: [redacted] State: [redacted] Zip Code: [redacted]

Home Telephone: [redacted] Cell Telephone: [redacted]

**STATE LAW REQUIRES WE HAVE TO DISCLOSE YOUR NAME UNLESS WE CAN SHOW THAT DISCLOSURE WILL RESULT IN SUBSTANTIAL HARM TO YOU, SOMEONE ELSE OR THE PUBLIC PER A.R.S. § 41-1010. IF YOU HAVE REASON TO BELIEVE THAT SUBSTANTIAL HARM WILL RESULT IN DISCLOSURE OF YOUR NAME PLEASE PROVIDE COPIES OF RESTRAINING ORDERS OR OTHER DOCUMENTATION.*

C. **PATIENT INFORMATION (1):**

Name: Corona

Breed/Species: Maltese Mix

Age: 7 months Sex: Female Color: Cream

PATIENT INFORMATION (2):

Name: _____

Breed/Species: _____

Age: _____ Sex: _____ Color: _____

D. **VETERINARIANS WHO HAVE PROVIDED CARE TO THIS PET FOR THIS ISSUE:**

Please provide the name, address and phone number for each veterinarian.

Dr. Boutet
Dr. Miller
Dr. Hubert
20610 N. Cave Creek Rd.
Phoenix, AZ 85024

E. **WITNESS INFORMATION:**

Please provide the name, address and phone number of each witness that has direct knowledge regarding this case.

- 3 Veterinarian Techs employed by VetMed Arizona whose names where not disclosed.
- Dr Stephanie Foote
VetMed AZ 20610 N. Cave Creek Rd Phoenix AZ 85024 602-697-4694
- Dr Alexandra Brower
Midwestern University [redacted]

Attestation of Person Requesting Investigation

By signing this form, I declare that the information contained herein is true and accurate to the best of my knowledge. Further, I authorize the release of any and all medical records or information necessary to complete the investigation of this case.

Signature: _____

Date: _____

F. ALLEGATIONS and/or CONCERNS:

Please provide all information that you feel is relevant to the complaint. This portion must be either typewritten or clearly printed in ink.

See Attached.

Background:

Owner: David and Cathy Evers

Name: Corona

Dog Breed: Maltese Mix

Age: 7 months

Sex: Female

Weight: 13.8 lbs

During the spay procedure in early July 2020 the attending veterinarian called to state that Corona had a heart murmur and would like to have it looked at by a veterinarian cardiologist prior to the spay procedure.

After numerous calls we were able to find and arrange for an appointment with the cardiology department at VetMed Arizona on July 21, 2020.

July 21, 2020:

On July 21, 2020 we brought Corona to VetMed Arizona for a Cardiopulmonary examination. The results showed that Corona had a left to right shunting PDA. Although, Corona is small a catheter procedure is recommended to deploy a device across her ductus. (Report is attached.)

Dr Boutet and a resident came out and spoke with Cathy and I about the finding and stated that Corona needed to have the procedure within the next 2 weeks. We discussed with him that we were scheduled to leave for Colorado for the month of August. He reiterated that the procedure needed to be completed as soon as possible. That we could take her to Colorado following the procedure, but that she would need to be sedated and kept quiet for 30 days for her recovery as this was the most risky time.

Dr Boutet stated that the procedure has a 98% success rate and that he "loved to do these procedures". Corona was young and in good health otherwise, so he expected a great outcome and this procedure would give her an otherwise healthy life with no restrictions after recovery. In fact, while he was going to be on vacation, he would happily come in to just do this procedure on Corona. Dr. Boutet stated he would have someone send us an estimate of costs and if we agreed he would schedule the procedure for July 28, 2020.

The following day VetMed Arizona e-mailed an estimate for the procedure. (attached). This estimate was not signed and returned to VetMed Arizona.

July 27, 2020:

Cathy and I dropped off Corona at approximately 6:00 PM at VetMed Arizona. At that time, the tech that took Corona asked if we wanted CPR performed just in case something went wrong. We told her yes.

July 28, 2020:

11:30 AM - Dr Boutet called and said that they were going to start the procedure and that it would take about 2 hours.

1:30 PM - Called and said the device didn't open like usual but it appeared ok. They would now start to wake up Corona.

2:00 PM - Dr Boutet called and said that Corona had liquid coming out of her air tube and looked to have aspirated. She had a heart attack and they were doing CPR. Asked if we wanted them to continue with CPR. Told Dr. Boutet yes that we wished him to continue. Dr Boutet said he would go back and tell them to continue. Numerous times during the conversation Dr Boutet stated" this shouldn't happen, this never happens"

2:10 PM - Dr Boutet called and stated they had got Corona's heart started but she had not awoken.

2:20 PM - Dr. Boutet called and said that Corona's heart had stopped again and do we want to continue with CPR. Cathy and I told him no and to let her pass on her own unless she appeared to be in pain. He said that she was not awake and was not in pain.

2:30 PM - Dro Boutet called and stated that Corona had passed away. He suggested that they would cover the expense of an autopsy and asked if we would like her sent to Midwestern University for an autopsy. We told him yes, that we would like to know what happened. He again during the conversation stated "this just doesn't happen".

August 8, 2020:

Called VetMed at 8:15 AM to see if the autopsy had been received. Was transferred to Zac (tech) in cardiology. He stated that as of August 4, 2020 the autopsy was still pending. He said that Dr Boutet was not in that day but would leave him a message to check on the autopsy. Asked Zac about a written report on the procedure. He stated that he did not see anything in Corona's file at that time. He did see the referral to Midwestern University.

I asked Zac who would have been in the room during the procedure. He said that he was not there that day but that normally there would be 3 cardiologist, 1 scrub tech, 1 tech doing echo and 1 other tech.

August 13, 2020:

Dr Boutet called to discuss the autopsy from Midwestern University (attached). He stated that it was inconclusive as to what actually happened to Corona but that it could have been a reaction to the contrast used. He would talk with the Dr. that performed the autopsy and get back to any additional information he could find out.

Late August, 2020:

Sometime during late August Dr. Boutet called and said that he had spoken with the Dr. at Midwestern University for about and hour and a half about the autopsy. The best that they could surmise was that Corona had either an allergic reaction to the contrast or that her blood pressure had spiked causing the hemorrhaging. He doubted the spike in blood pressure was the issue as that is monitored during the procedure.

August 31, 2020:

We received a message from Katie at VetMed Arizona that she needed our credit card number to process a refund. Upon speaking with Katie she stated that a $525.37 refund was due to us. I asked what this pertained to. She said there was nothing specific but that Dr. Boutet had authorized a refund. I asked if it was due to Corona's death. Katie said "pretty much". I asked to have Dr. Boutet call me as a 10% refund was not sufficient for the death of our puppy.

September 3, 2020:

Spoke with Dr Boutet twice concerning the refund of the cost of the procedure. He stated that he could only authorize the refund of soft costs (totaling approximately $1,600). That any hard costs would need to be approved by Stephanie Foote, the medical director for VetMed Arizona. Asked Dr Boutet to please have Ms Foote to give me a call.

September 6, 2020:

Dr Foote called. I asked her to please refund the cost of the PDA procedure as something happened in that room that resulted in the death of Corona. She stated that that is the risk that we take in any procedure whether it is a PDA procedure or setting a broken bone. Told Dr. Foote that Dr Boutet had specifically stated that the procedure had a 98% success rate and that the risk was in the recovery. Asked Dr Foote if VetMed Arizona had ever had a death from this procedure. She stated that there had been no previous deaths. Told her that Dr Boutet had already offered a refund of approximately $1,600. She stated that we were aware of the risks and she would not refund anything else. In that case I asked her to please inform the 3 doctors in the procedure that we would be filing a complaint with the Arizona Veterinary Board.

Complaint:

Against:

- Dr Bruno G Boutet
- Dr Mathew W Miller
- Dr Sage H Hubert
- VetMed Arizona

Arizona Revised Statutes Title 32, Chapter 21

Article 3 32-2232 Unprofessional or dishonorable conduct:

22: Medical incompetence in the practice of veterinary medicine.

- Death of Corona in a procedure that was touted by Dr Boutet as a 98% success rate, industry reports show a 98 to 100% success rate and Dr Foote stated that VetMedAZ had never lost a patient to this procedure.

- Dr Boutet stated that Corona may have had an allergic reaction to the contrast. The estimate provided by VetMedAZ prior to the procedure showed one injection of Iohexol. Due to the ACDO not opening as normal an additional injection of contrast was administered. This may have been an overdose of contrast.

- As documented in the surgical report and communicated by Dr. Boutet during the procedure, the ACDO did not open as normal. This may have been failure to insert or open the device as required. This would be bordering on gross negligence in performance of the procedure.

21: Failure to maintain adequate records of veterinary services provided:

On September 10, 2020 we requested a copy of all records for Corona. On September 11, 2020 those records were e-mailed to us by Allicia at VetMedAZ. The records received are attached.

- The surgical report dated July 28, 2020 fails to report the following:
  - Who did the procedure
  - Who other than the 3 doctors were in attendance
  - Who administered and how was anesthesia administered.
  - Amount of contrast injected into Corona
  - Communication with the owners of Corona (4 individual calls by Dr Boutet)
  - NO MENTION THAT CORONA DID NOT SURVIVE OR WHAT HAPPENED.
    - What actions were taken to resuscitate Corona
    - Who performed those actions
    - What, if any, drugs were used.

Requested Actions:

1. Return of all fees paid for the procedure that resulted in the death of Corona. ($5,057). Dr Boutet has already offered to refund approximately $1,600 due to Corona's death.

2. Suspend all 3 Drs ability to perform the PDA catheterization procedure until they have completed additional formal education on the procedure.

3. Restrict all 3 Drs from touting the PDA procedure as having a 98% success rate unless they also disclose in writing that they have personally overseen a procedure that resulted in the death of a patient.

4. Suspend the license of Dr Boutet until he has completed ethics training for failure to maintain adequate records of veterinary service provided.

Despite 3 Drs and 3 others (undisclosed) being in the room during the procedure, something happened that resulted in an otherwise healthy puppy losing its life. No one is able to explain the reason or why she lost her life. There are many questions that are unanswered and only those individuals in attendance actually know the circumstances and actions taken that resulted in Corona's death. The lack of record keeping and failure to provide the names of the other individuals in attendance, leads us to believe that some action was taken that directly caused the death of Corona.

Article

Mar 8, 2019

Patent Ductus Arteriosus in Dogs

Cardiology

September 2010 (Vol 32, No 9)

- by
- Kristyn D. Broaddus , DVM , MS , DACVS ,
- D. Michael Tillson , DVM , MS , DACVS

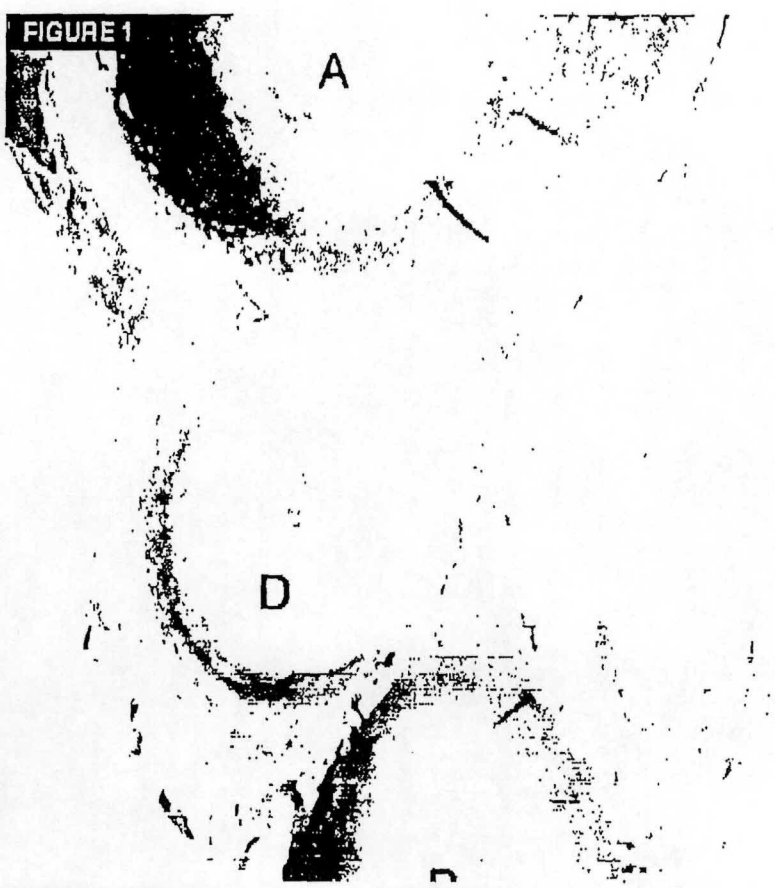
Abstract

Patent ductus arteriosus (PDA) is the most common congenital heart disease in dogs. It is due to the failure of the ductus arteriosus muscle to constrict, leaving a passageway for blood flow and resulting in eventual left-sided heart disease and/or generalized heart failure. It is hereditary in several breeds. The typical left-to-right PDA is amenable to minimally invasive procedures or open surgery. The ideal surgical candidate for PDA occlusion is immature and lightweight, with minimal heart changes. There is a wide variety of surgical techniques involving different methods of dissection and suture passage. Intraoperative hemorrhage during dissection is the most serious potential complication and can be life-threatening. Minimally invasive techniques such as thoracoscopic ligation and intravascular coiling have been claimed to have lower morbidity and mortality than open techniques. Once the PDA is occluded, most patients have remodeling of the myocardial tissues, resulting in an excellent long-term prognosis. Late complications such as residual flow and recanalization are rare but may be clinically significant.

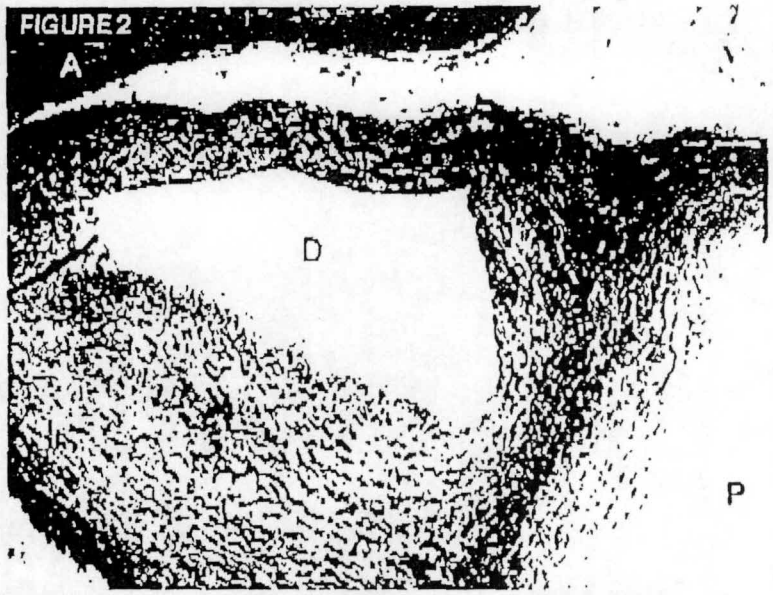
The first surgical correction of a human patent ductus arteriosus (PDA) was performed in 1938.[1] Less than 2 decades later, Dr. Willis Potts was the first to perform surgical ligation of a PDA in a dog.[2] Today, PDA is recognized as one of the most common congenital heart defects in dogs, with an incidence ranging from 25% to 30% of cases.[3,4] Female toy-breed dogs are overrepresented in this condition. A characteristic history and clinical signs, along with a classic "machinery" murmur, typically lead to procedures such as chest radiography

The ductus arteriosus (DA), also referred to as the *arterial duct*, *arterial canal*, and *ductus Botalli*, forms the sixth aortic arch.[3] The DA extends from the bifurcation of the main pulmonary artery to the ventral aspect of the descending aorta between the left subclavian artery and the intercostal arteries. It normally comprises 98% smooth muscle, with subadventitial elastic fibers and loose collagen intermingled within the adventitia.[3] In the fetus, the DA shunts blood away from the nonfunctional lungs back to the systemic circulation. At this time, the fetal lungs are collapsed and pulmonary vascular resistance is suprasystemic, allowing only 5% to 8% of blood flow from the pulmonary artery to reach the lungs.[8] Fetal blood is oxygenated instead by the placenta. The presence of the DA also allows the right side of the heart to grow and develop.

At birth, the neonate's lungs expand. This allows dilation of the pulmonary arterioles and a profound reduction in the pulmonary vascular resistance to approximately 20% of the systemic resistance. Pulmonary vascular resistance is further diminished by thinning of the smooth muscle within the pulmonary arterioles.[8] During this time, an increase in systemic oxygen tension stimulates the smooth muscle within the DA to constrict in a process called *apobiosis*.[3] Diminished circulating prostaglandin also plays a role in DA closure. In utero, circulating prostaglandin levels are high due to placental production and minimal pulmonary metabolism. At birth, the placenta no longer serves as a source of prostaglandins, and prostaglandin metabolism by the lungs increases. With the inhibiting influence of prostaglandins dwindling, the DA is able to close. The closed DA predominantly comprises uniform, circumferential smooth muscle cells and very little elastic tissue (**FIGURE 1**).[3] Physiologic closure of the DA occurs immediately after birth; anatomic closure follows within a 48 hours to 1 month.[9] By 1 month of age, the muscle cells degenerate via cytolysis and the DA becomes a nonpatent elastic structure referred to as the *ligamentum arteriosum*.[8]

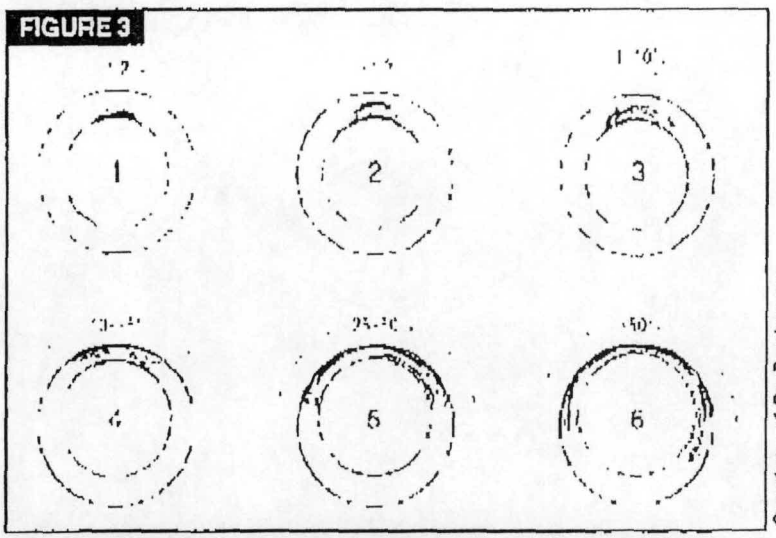


that is generally funnel-shaped, with the narrowest portion adjacent to the pulmonary artery.[3] Typically, a fibrous ridge of incomplete ductus muscle within the PDA lumen incompletely narrows the orifice.[3] A system has been developed for the histologic grading of PDA based on the amount of abnormal elastic tissue present (**FIGURE 3**). The severity of the grade increases with the proportion of defective genome inherited from affected parents.[10] In one study, dogs with large PDAs that had a reversal of flow typically had the smallest amount of smooth muscle within the PDA wall (i.e., a grade 6 PDA) and conversely had the greatest amount of elastic tissue in the wall.[3]



Courtesy of Dr Buchanan

Transverse histologic section of a PDA (*D*) and adjacent aorta (*A*) and pulmonary artery (*P*) in an 11-day-old dog with a grade 5 PDA. The ductus muscle (*DM*) is asymmetrically constricted. The portion adjacent to the aorta is not constricted and has a thicker elastic segment.



Courtesy of Dr Buchanan

PDA grading system based on the extent of aorta-like elastic tissue (*shaded area*) in the normally muscular PDA wall.

The PDA courses within the wall of the aorta before emptying into the aortic lumen, forming an aortoductal aneurysm.[3] Generally, the size of the aortoductal aneurysm varies inversely with the length of the ductus: the shorter the surgical segment, the larger the aneurysm and

atrium, and left ventricle. Because this volume overload is chronic, the left side of the heart undergoes eccentric hypertrophy. Eventually, this left-sided overload leads to left-sided CHF that can become generalized as the right side of the heart continues to pump blood through a fibrotically stenosed pulmonary vasculature to a decompensating left heart.[8] In a patient with a PDA, this can occur as early as 1 week of age or may develop many years later; however, 70% of dogs with PDA develop clinical signs of CHF before 12 months of age.[5] The timeline for the development of CHF appears to depend on the diameter of the PDA. A dog with a small PDA may not display clinical signs of CHF until later in life. There is a report of a 15-year-old cocker spaniel with a small, incidental PDA identified at necropsy.[13] In the worst cases, a large PDA may allow bidirectional or right-to-left shunting within the first month of life.[3]

Heritability

Although several early epidemiologic studies suggested a genetic component to the occurrence of PDA, in 1971, Patterson et al[10] documented that PDA in toy and miniature poodles is "a specific, localized, developmental anomaly which is genetically determined." This mode of inheritance does not follow a simple Mendelian pattern. When normal dogs were crossed with dogs that had PDA, some offspring had PDA whereas others had an intermediate condition in which the DA closed only at the pulmonary arterial end to create a ductus diverticulum. This finding suggests that the trait is quasicontinuous, that is, a threshold trait with graded phenotypic expression. In other words, the chance of a dog having PDA and the severity of the abnormality increase with increasing amounts of defective genome from the parents. The results of these breeding experiments supported a two-threshold model of inheritance. When the first threshold is reached, a partial closure of the DA results in a ductus diverticulum. If the second threshold is reached, a PDA results. The lowest incidence of defective DA closure was noted in the offspring from normal dogs bred with dogs with apparent PDA (20%). An intermediate incidence was noted in the offspring when unaffected dogs with affected first-order relatives were mated with dogs that had ductus diverticula. The highest incidence (80%) of PDA was noted in offspring when both parents had PDA.[10]

Buchanan and Patterson[14] found similar structural abnormalities in sporadic cases of PDA in collies, cocker spaniels, German shepherds, Pomeranians, Shetland sheepdogs, and shih tzus, suggesting that there may be a similar genetic component in these breeds. A genetic basis for the occurrence of PDA has also been documented in Welsh corgis.[4] This further supports the conclusion that dogs with a PDA should not be bred, even if the breed is atypical for the anomaly.

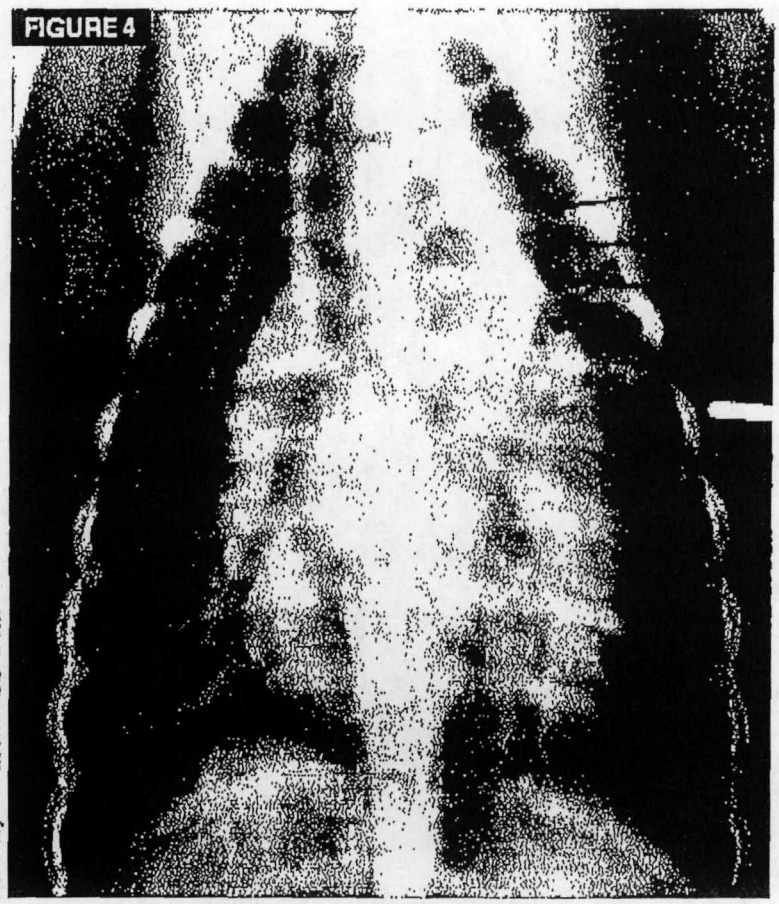
Presentation and Clinical Signs

PDA is overrepresented in female toy and miniature poodles; Maltese; Pomeranians; Shetland sheepdogs; cocker and English springer spaniels; keeshonden; bichons frises; Yorkshire terriers; and collies.[4,5] When identified at a young age, dogs may have no clinical signs or may present with mild exercise intolerance and stunted growth. Owners may mention that they can feel the puppy's heart "buzzing." Many dogs with PDA are identified when they present for routine puppy vaccinations at 6 to 12 weeks of age, when a left-sided, continuous machinery murmur is auscultated. In most cases, a palpable thrill is noted.

gradient between the systolic and diastolic pressures.[8,15,16] This occurs because of diastolic "run-off" through the PDA, causing a lower-than-normal diastolic pressure. A very small PDA may not have a palpable thrill.

Thoracic radiography is useful for evaluating the anatomic changes consistently seen in patients with PDA. Dorsoventral and lateral views of the thorax should be obtained for proper cardiopulmonary evaluation. The larger the PDA or the older the patient at the time of evaluation, the more prominent the radiographic changes will be. Specific changes include progressive enlargement of the left atrium, left ventricle, aortic arch, and pulmonary arteries. The dorsoventral view is helpful for evaluation of the heart and great vessels. The cardiac silhouette may appear elongated due to aortic arch enlargement cranially and left ventricular enlargement that extends the silhouette caudally. The right apical lung lobe vessels are considered enlarged if they are greater in diameter than the smallest diameter of the fourth rib. Extreme cardiomegaly may shift the heart into the right hemithorax. The most characteristic sign of PDA is an aneurysmal bulge in the aorta at the level of the ductus.[3] This "ductus bump" can be seen radiographically as a lateral deviation of the left lateral wall of the descending aorta at the level of the main pulmonary artery (**FIGURE 4**); however, this change is not always present. On the lateral view, overperfusion of the lungs can be appreciated, as well as left-sided heart enlargement indicated by dorsal elevation of the trachea and increased sternal contact (**FIGURE 5**).

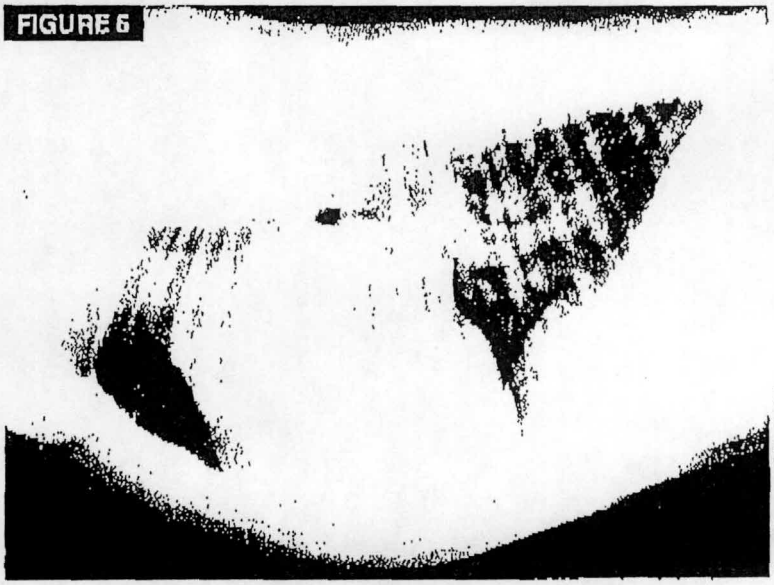
FIGURE 4



Courtesy of Jason Arble, DVM

Dorsoventral view of the thorax in a patient with PDA. The cardiac silhouette is elongated due to left-sided heart enlargement. Note the ductal aneurysm (*arrows*).

FIGURE 6



Lateral view of a patient with a PDA. Note the increased height and width of the cardiac silhouette, increased sternal contact, and dorsal elevation of the trachea. The pulmonary vasculature is overperfused, and there is evidence of early pulmonary edema.

Electrocardiography (ECG) should be conducted to evaluate heart chamber enlargement. Tall R waves (more than 2.5 mV) or wide P waves are typically noted on a lead II tracing. In patients with advanced heart disease from PDA, atrial fibrillation or ventricular ectopy may be noted. Atrial fibrillation is a late change and is associated with a grave prognosis.[6] It is the result of an incompetent mitral valve that allows such significant backflow of blood that the left atrium is severely stretched.

Echocardiography may not always be required as part of the workup of a suspected PDA, but it may confirm the diagnosis and help to identify any other cardiac defects. It can also demonstrate changes in cardiac wall thickness and chamber size. To visualize the PDA, the right parasternal short-axis and left cranial window views are most useful.[12] Identification of PDA often involves finding a high-velocity, turbulent flow pattern in the pulmonary artery as the blood exits the PDA. This is best detected using color-flow Doppler imaging. The degree of shunted blood flow is reflected in the magnitude of left atrial and ventricular eccentric hypertrophy.[12] The left ventricular outflow ejection velocity is normally increased along with transaortic and transmitral flow velocities. Increased ejection volumes may result in flattening of the intraventricular septum. Identification of PDA with echocardiography depends on the skill of the individual performing the examination. Nuclear scintigraphy can also be used to quantify left-to-right and right-to-left shunts.[17,18]

Right-To-Left Shunts

A small percentage of patients with PDA present with severe exercise intolerance and/or pelvic limb collapse during exercise. These patients have a reversal of normal flow through the PDA that can be documented with color-flow Doppler imaging. This reversal mixes nonoxygenated blood from the pulmonary artery with oxygenated blood from the aorta. The hallmark of a right-to-left shunt is differential cyanosis of the caudal half of the body. This is best visualized on examination of mucous membranes. Cyanosis can occur cranially as well

hypertrophy, which slowly increases local vascular resistance. These arteries become damaged from persistent pressure, and growth factors become elevated, leading to smooth muscle cell hypertrophy/hyperplasia and connective tissue protein synthesis. The resultant medial hypertrophy and intimal proliferation lead to progressive narrowing and increasing pulmonary vascular resistance. In dogs with large PDAs, between 3 months and 3 years of age, pulmonary vascular resistance exceeds systemic vascular resistance and blood flow reverses, creating a right-to-left shunt. The distinctive PDA murmur is lost during this transition.[8]

A right-to-left PDA becomes clinically important when it shunts a large amount of nonoxygenated blood from the pulmonary circulation (pulmonary artery) into the systemic circulation (aorta). This can reduce circulating arterial oxygen tension to a level typically between 30 and 45 mm Hg (normal: ~100 mm Hg). Because the subclavian artery and brachycephalic trunks branch off of the aorta cranial to the PDA, most of the mixing of nonoxygenated and oxygenated blood occurs in the descending aorta, resulting in inadequate oxygen delivery to the caudal portion of the body. Exercise exacerbates caudal cyanosis by decreasing systemic vascular resistance. In the meantime, the body attempts to compensate for worsening oxygen deprivation and chronic renal hypoxia by triggering the release of erythropoietin. The resulting polycythemia is a helpful adaptation when the hematocrit is 55% to 65% but becomes detrimental at higher hematocrit levels.[8] Eventually, the blood becomes hyperviscous, slowing the circulation and further impairing oxygen delivery. The pulmonary vasculature responds through continued vasoconstriction, which perpetuates the pulmonary hypertension.[8]

Treatment and Outcome

Interventional Therapy

PDA in dogs can be managed with medical therapy or occlusion using either open surgical ligation or minimally invasive techniques. In human pediatric medicine, nonsurgical occlusion of PDA is accomplished by using prostaglandin synthase inhibitors (e.g., indomethacin, ibuprofen) to stimulate natural closure.[19] The use of prostaglandin synthase inhibitors does not appear to be effective when there is hypoplasia of the smooth muscle of the DA, however, which is the most common scenario in dogs.[3] Moreover, dogs are generally diagnosed with PDA several weeks to months after birth, when the smooth muscle within the DA is no longer responsive to antiprostaglandin therapy. Due to the ineffectiveness of medical intervention, mechanical occlusion of the PDA remains the mainstay of treatment in dogs.

Since the 1950s, when the first surgical repair of canine PDA was performed, surgical ligation has proved to be an effective procedure in the proper candidates. The ideal canine patient for surgical closure of a left-to-right shunting PDA is a dog between 8 and 16 weeks of age with no concurrent cardiac disease and minimal secondary heart changes. Contraindications to occlusion of a PDA are right-to-left shunting, bidirectional shunting, or concurrent cardiac conditions that rely on the PDA for survival (e.g., tetralogy of Fallot). Older dogs with a recent diagnosis of a hemodynamically significant PDA should undergo surgery as soon as possible if they are reasonable surgical candidates.[16] Severe secondary myocardial failure does not

hypoglycemia. A jugular catheter is useful for blood draws and rapid infusions of fluids or blood products and may be appropriate in high-risk surgical patients. Preoxygenation can be used before anesthesia induction. Patients should be premedicated with an opioid. I do not routinely premedicate these patients with anticholinergics, giving these drugs only if the heart rate drops below acceptable levels; however, routine premedication with anticholinergics has been advocated.[20] Anesthesia is induced by IV injection of an ultrashort-acting barbiturate (e.g., thiopental, propofol) or inhalation of gas via face mask. Etomidate, a carboxylated imidazole derivative, may be a more favorable induction agent for patients with CHF.

Surgical Ligation

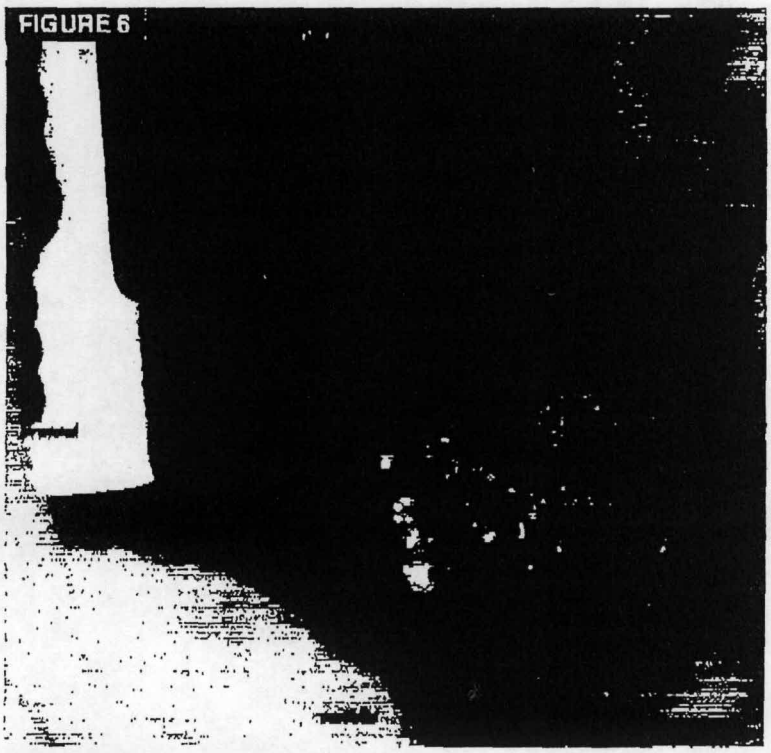
The first surgical ligation of a PDA in a dog was recorded in 1952. Surgical interventional techniques have not varied significantly since 1967, when a detailed surgical report of a successful PDA ligation in a 4-week-old male mongrel puppy was published.[21]

The standard approach for PDA ligation in dogs remains a left fourth intercostal thoracotomy. The patient is positioned in right lateral recumbency, with a small rolled towel placed under the cranial thorax to maximize exposure by arching the chest and spreading the ribs on the left side.[22] The forelimbs may be secured in gentle extension. The patient's entire thorax should be clipped and prepared just beyond the dorsal and ventral midlines, extending cranially to the point of the shoulder and caudally to the last rib. It is helpful to clip the caudal aspect of the proximal antebrachium, including the elbow, as it is often in the surgical field. The skin incision is centered by counting the intercostal spaces back from the palpable 12th space. A generous, curved skin incision is made from just ventral to the vertebral processes to ventral to the costochondral junction along the desired intercostal space. The incision is continued down through the subcutaneous tissue and cutaneous trunci muscle. The latissimus dorsi muscle is sharply incised along the same line, although some surgeons prefer to retract the latissimus muscle dorsally, which may decrease the postoperative discomfort associated with muscle transection but may also limit visualization.

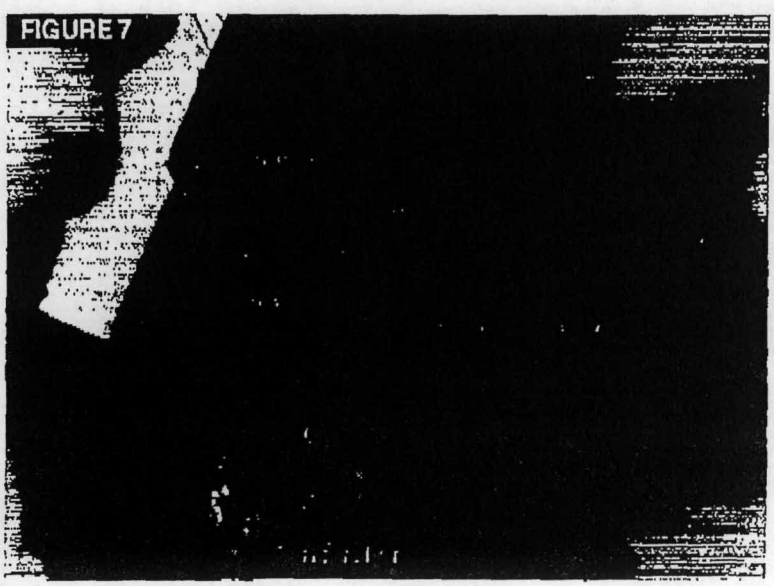
Once deep to the latissimus dorsi, the surgeon should recount the intercostal spaces by palpating the first rib and counting caudally to the proposed incision site. The ventrally located scalenus muscle serves as a landmark for a fourth intercostal incision because the muscular portion of the scalenus inserts on the fifth rib, with fibrous bands extending the insertion to the eighth and ninth ribs. The scalenus muscle is incised over the fourth intercostal space, and the serratus ventralis muscle fibers are separated and retracted dorsally or sharply incised. The incision through the external and internal intercostal muscles is centered between the ribs to avoid the intercostal vessels running in the costal groove on the caudal aspect of the ribs. Metzenbaum scissors are used to lift the muscles away from the pleura and to minimize risk of iatrogenic injury to the underlying lungs.

Once the pleura is identified deep in the intercostal muscles, it should be carefully penetrated with scissors during exhalation. This creates a pneumothorax, allowing the lungs to fall away from the incision. The patient must be manually ventilated at this time. The pleural incision is carefully extended dorsally and ventrally. When the incision reaches the costochondral junction, care must be exercised to avoid injury to the internal thoracic artery where it courses subpleurally along to the sternum. Maintained laparotomy sponges or 4 × 4 gauze

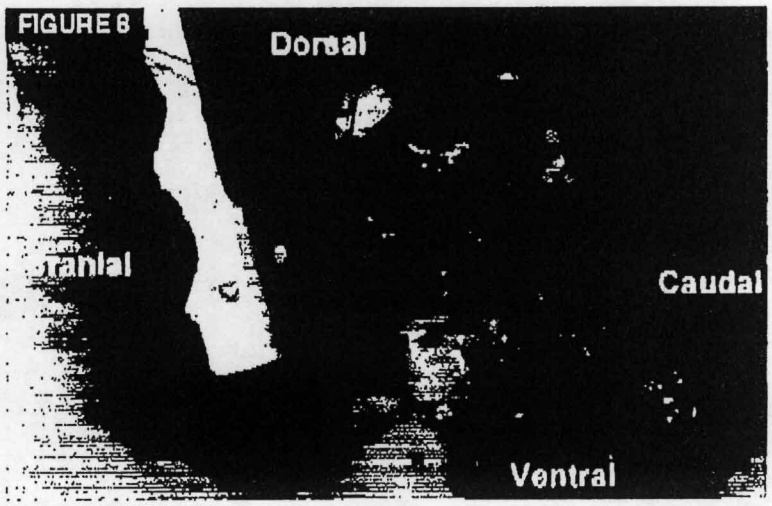
ductus is dissected bluntly without entering the pericardium. Using right-angle forceps or Lahey bile duct forceps, dissection is initiated parallel to the caudal aspect of the ductus. The tips of the forceps are inserted in a closed position and slowly opened to gently separate the tissues surrounding the PDA. The right-angle forceps are withdrawn with the jaws partially open. Closure of the forceps while in the tissues could result in inadvertently grabbing or tearing the fragile wall of the ductus. Once a lateral-to-medial dissection plane is opened on the caudal aspect of the ductus, a cranial dissection plane can be established. This dissection plane is created by angling the right-angle forceps approximately 45° and using the same technique of gentle dissection and withdrawing the forceps with the jaws open. Due to the difficult angle of cranial dissection, straight or curved forceps or hemostats are sometimes used.



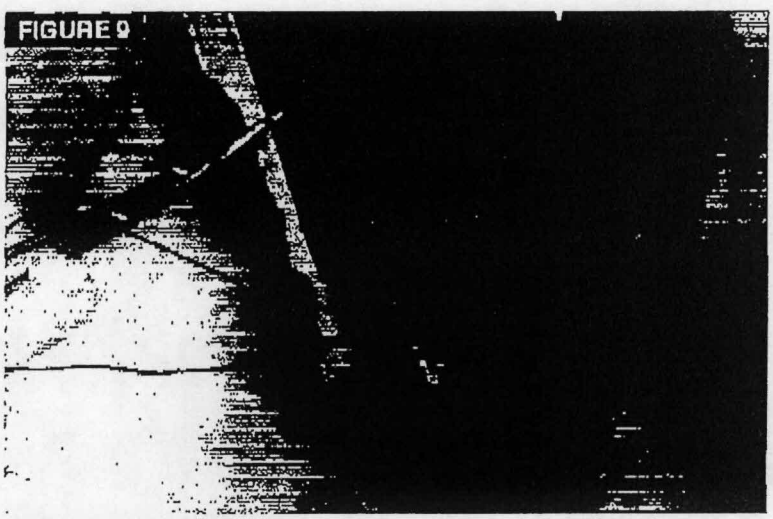
Intraoperative view of the heart at the fourth intercostal space. The vagus and phrenic nerves are visible. Note the vagus nerve as it courses over the ductus.



cranial dissection plane. Inexperienced surgeons are cautioned to exercise patience at this point and to continue the slow, steady dissection on the medial aspect. Some surgeons recommend using a moistened cotton-tip swab to aid in dissection.[23] Utmost caution is necessary at this point because most episodes of hemorrhage caused by a ductal tear occur during the medial dissection of the ductus (**FIGURE 8**). When the tips of the right-angle forceps can be safely opened cranially, a strand of suture is introduced into the jaws for passage around the ductus. A pliable, heavy, nonabsorbable suture material (e.g., 1, 0, or 2/0 silk or Dacron) is recommended for ductus ligation (**FIGURE 9**). The ductus is closed by double ligation; the surgeon can either pass two separate strands of suture material or create and pass a loop of a single strand that is then cut to yield two pieces (**FIGURE 10**).

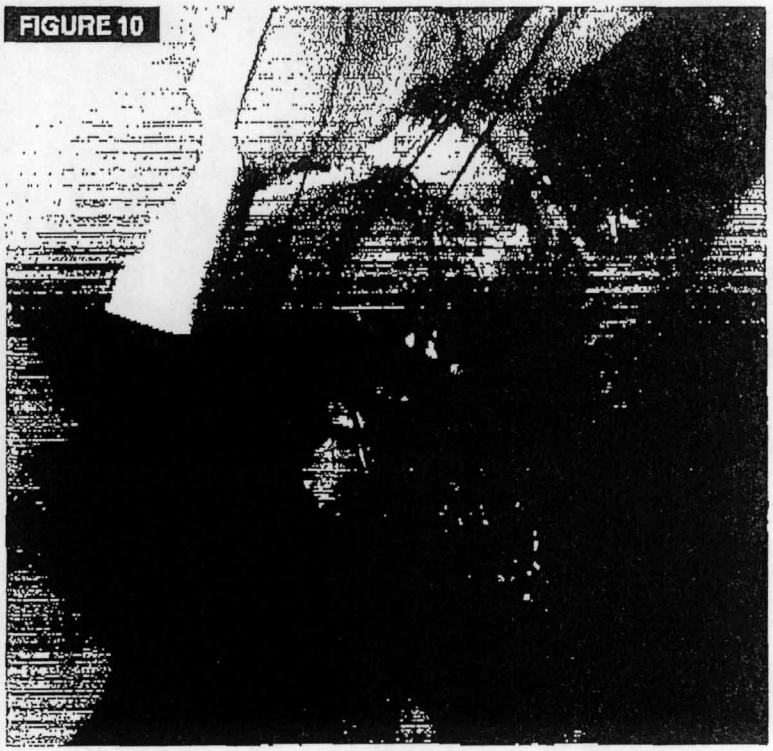


Intraoperative view of a PDA. The vagus nerve is gently retracted dorsally with suture. Right-angle forceps are seen passing caudally to cranially; the tips can be seen exiting cranially. The phrenic nerve can be seen ventral to the PDA.



Intraoperative photograph of a suture loop (0 silk) passed around the medial aspect of the ductus. The loop will be transected to provide two pieces of suture to be tied separately.

FIGURE 10



Once the loop of suture is transected, two strands are separated. One is retracted toward the aorta (upper strand) and the other is retracted toward the pulmonary artery (lower strand). The strand closer to the aorta is tied first.

With either technique, the surgeon must be careful not to cross the suture strands on the medial aspect of the ductus. Also, the surgeon should never force the passage of the suture material around the ductus. If the suture does not pass smoothly, the forceps are opened, the suture is released, and then the forceps are withdrawn and replaced for another attempt at passage. Patience and adequate dissection around the medial aspect of the ductus will ultimately ease the passage of the suture material.

Once two strands of suture have been passed, they are checked to ensure that they are not entwined. The suture material should slide freely around the ductus but should not be aggressively manipulated, which can cause the suture to erode through the ductus wall, resulting in catastrophic hemorrhage. When the surgeon is ready to occlude the ductus, the sutures are tied. The suture closest to the aorta is ligated first. The ligature is slowly tightened, and the knot is secured with a minimum of five throws. The patient may develop a drop in heart rate at this time (i.e., Branham sign), a reflex bradycardia due to a sudden increase in aortic pressure as the PDA is ligated. Some authors recommend attenuating the ductus over a period of 2 to 3 minutes to minimize this effect.[24,25] In patients that experience significant bradycardia, an anticholinergic drug (e.g., atropine, glycopyrrolate) can be given.[5] While the Branham sign is not seen with every PDA ligation, the correct dosages of anticholinergics should be readily available. I (K. D. B.) do not routinely use anticholinergics as part of premedication or during surgery unless the patient's heart rate drops below 50 bpm. In most cases of postligation bradycardia, the heart rate recovers in a few minutes.

If the patient remains hemodynamically stable, the suture strand on the pulmonary artery side is tied. The ductus and pulmonary artery can be palpated for the absence of a turbulent

Chest wall closure is begun using four to eight evenly spaced circumcostal sutures that are placed by skimming the cranial aspect of the fourth rib and taking a slightly larger bite of tissue around the caudal edge of the fifth rib. The type and size of suture material depend on surgeon preference. I (K. D. B.) use absorbable sutures (e.g., PDS II, Ethicon) in sizes ranging from 1 to 3/0, depending on patient size. Other surgeons may prefer using an appropriately sized nonabsorbable suture. An assistant (if available) crosses two adjacent sutures to appose the ribs and allow the surgeon to tie the initial suture. This process is repeated until the ribs are securely apposed. Alternatively, a rib approximator can be used to appose the ribs, taking care to avoid overlapping them. A technique of placing transcostal sutures (i.e., suture passed through holes drilled in the body of the rib) has been described to avoid entrapment of the intercostal nerve on the caudal surface of the rib in the circumcostal suture, which can cause postoperative pain.[26] This report did not discuss the biomechanical effects of drilling multiple holes in the ribs. The serratus ventralis and scalenus muscles are then sutured back to achieve one layer of soft tissue closure. The incised edges of the latissimus dorsi muscle are reapposed or released from their retracted position.

Once an airtight seal is achieved, the thoracic cavity is evacuated via a butterfly catheter, needle, or chest tube with a three-way stopcock for easier aspiration and disposal of air and fluid. The panniculus, subcutaneous tissue, and skin are closed routinely. I (K. D. B.) prefer to place skin sutures in toy-breed puppies, but an intradermal suture pattern or skin staples can be used, although these may not engage well in smaller animals. With the patient still under anesthesia, a light wrap may be placed over the thoracic incision to avoid trauma to the incision and provide light support to the thorax. The wrap includes a nonadherent contact layer, an absorbent layer, and an outer protective layer. Great care must be taken to ensure the thoracic wrap does not impinge on the respiratory excursions of the patient and cause dyspnea, hypoxia, and cyanosis. An excessively tight thoracic bandage could result in the death of a small patient in the postoperative period. Most patients are discharged approximately 48 hours after PDA ligation surgery with a loose "t-shirt" or stockinette covering the thorax.

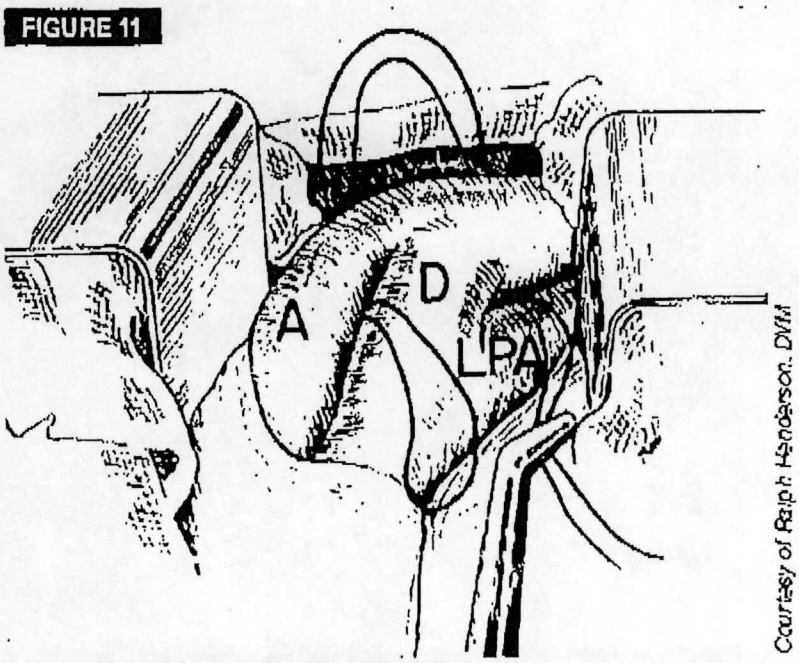
Variations in Surgical Technique

Several variations on dissection and ligation technique and suture passage exist. Because the medial aspect of the PDA is potentially weak and catastrophic hemorrhage is possible, sterile cotton swabs can be used for dissection of the cranial and caudal aspects of the PDA in lieu of forceps.[23] As for variations on suture passage, one author recommends using right-angle forceps to pass a knotted double strand of suture with the loop cut off. This technique is meant to avoid engaging soft tissue from the blind side of the PDA by preventing complete closure of the jaws of the forceps, which are forced slightly apart by the knot.[27] Another technique uses a stainless steel orthopedic wire loop (18- to 20-gauge) passed from caudal to cranial to safely pull suture around the medial aspect of the ductus.[28]

Perhaps the second most commonly used suture passage technique is the Jackson-Henderson method. This technique is designed to avoid "tedious PDA dissection by drawing the ligatures from the dorsal and medial aspects of the aorta."[29] The aorta is dissected free from its mediastinal pleura between the left subclavian artery and first intercostal artery to create a space for suture passage. Right-angle forceps are placed cranial to the ductus around the dorsomedial aspect of the aorta. The suture loop is presented from this location and pulled

flow were not stated. Stanley et al[25] suggested that complete ligation is not achieved with the Jackson-Henderson technique due to excessive soft tissue inclusion in the ligatures. Bellenger et al[30] thought that this technique decreased the risk of hemorrhage but increased the risk of residual shunting. Other studies showed no adverse effects of the Jackson-Henderson technique on patient outcome.[31,32]

FIGURE 11



Courtesy of Ralph Henderson, DVM

Illustration of the Jackson-Henderson technique of suture passage around the PDA. The suture is passed around the descending aorta on either side of the ductus. This avoids direct dissection around the medial aspect of the PDA.

In 1971, Breznock et al[11] reported on the use of tantalum hemostatic clips for the ligation of PDA to avoid medial dissection and facilitate application. One large (10-mm) tantalum clip was placed over the ductus and compressed in as little as 5 minutes with no intraoperative hemorrhage attributable to the ductus.[11] These clips close from the tip toward the body, supposedly avoiding any extrusion of the PDA vessel. Tantalum clips are rarely used in humans due to the risk of recanalization.[11] More recently, the use of tantalum hemoclips was reexamined in a study of 20 dogs, 19 of which had successful complete occlusion of the PDA.[33] Medial dissection was avoided, but hemorrhage comparable to that associated with routine surgical ligation was noted at the cranial dissection site (10%). One dog had incomplete occlusion with persistent but minimal residual flow at day 560 postoperatively. This study did not report any recanalization and had a mean follow-up of 799 days.[33]

Surgical Complications

Overall, surgical complications are minimal for routine PDA ligation performed by an experienced surgeon. Mortality is reported at 0% to 2% for surgeons who have performed more than 100 such operations.[4] Complications include hemorrhage, laryngeal dysfunction, air embolization, central nervous system hypoxia, myocardial hypoxia, hypothermia, and hypercapnea/hypocapnea with subsequent respiratory acidosis or alkalosis.[34]

The most serious complication encountered is traumatic injury to the PDA. The occurrence of

an incision into the pericardium to give access to the base of the main pulmonary artery and ascending aorta.[24] Eyster[20] opens the pericardium routinely for better access to the PDA, pulmonary artery, and aorta for emergency cross-clamping. A tangential vascular clamp is placed across the pulmonary artery and aorta, cranial to the aorta and caudal in the pericardial sinus between the left atrium and pulmonary artery. The resulting ventricular outflow occlusion induces circulatory arrest. This can be maintained under normothermic conditions for up to 8 minutes.[24] The descending aorta can be digitally occluded to prevent further blood loss. The ductus is then dissected from the caudal aspect, exiting more cranially than the initial dissection. Ligation is completed quickly, and the vascular clamp is removed. Once the heart has adequately filled, digital pressure is discontinued on the descending aorta and a transfusion is rapidly initiated. The three dogs in which this technique was used survived without defibrillation or inotropic support, but these options should be available. This technique would not be effective on large tears.[24]

If the tear in the ductus is large and does not respond to tamponade, large vascular clamps can be placed and large, deep, biting mattress sutures placed in an attempt to occlude the ductus and control the hemorrhage. Another technique involves division of the PDA with oversewing of the ends. To do this, a large tangential vascular clamp (e.g., Satinsky clamp) is placed on the aortic side of the ductus and an angled 45° or 90° vascular clamp is placed on the pulmonary artery side. The ductus is transected, and each end is secured with a buttressed continuous mattress suture oversewn with a simple continuous pattern.[4] This technique is also recommended by some authors for PDAs that are greater than 1 cm in diameter and/or for aorticopulmonary windows.[22]

Several authors have described techniques to induce hypotension for the management of intraoperative hemorrhage. Nitroprusside has been used to lower blood pressure to 45 to 60 mm Hg to slow or stop ductal hemorrhage.[23] Its effects can be seen within 10 minutes of initiating the IV infusion. Nitroprusside and phentolamine have also been used before PDA dissection to lower pressures and decrease the severity of hemorrhage in the event of ductus trauma.[23]

Key Points

- Patent ductus arteriosus is the result of asymmetrical distribution of ductus smooth muscle, preventing complete closure of the ductus arteriosus.
- The aortic aneurysmal dilation may not resolve after successful ligation or occlusion of a patent ductus arteriosus.
- Patent ductus arteriosus is a heritable condition, and affected patients should not be bred.
- In the hands of an experienced surgeon, surgical complications should be minimal.
- Depending on the amount of defective genome that is inherited, manifestations range from an asymptomatic ductus diverticulum to a clinically significant patent ductus arteriosus.

Risk Factors for Surgical Complications

complications such as hemorrhage from the PDA negatively affected long-term survival.[31] In 2005, another study reported that age, weight, lethargy, preoperative treatment with angiotensin-converting enzyme inhibitors, and right atrial dilation on radiography were all negatively associated with survival.[6] In this study, 92% of dogs survived to 1 year and 87% survived to 2 years. No dogs died of heart-related disease beyond the second year after surgery. Forty-two percent of dogs in this study had mitral regurgitation, but this finding was not related to survival.[6] Recently, Eyster[20] reported that age and size are not factors in successful surgical treatment of PDA.

Minimally Invasive Techniques

Minimally invasive techniques for PDA occlusion have migrated into veterinary medicine as the fields of interventional radiology and cardiology and minimally invasive surgery have grown. Currently, minimally invasive procedures for PDA occlusion can be divided into intravascular techniques and thoracoscopic surgery. Intravascular procedures are described briefly here, but veterinarians interested in these techniques should undergo training with an experienced surgeon.

Intravascular techniques for PDA occlusion involve the use of either thrombogenic coils or intravascular occluding devices (duct occluders or vascular plugs). Thrombogenic or embolization coils (Cook Medical; Bloomington, IN) are composed of surgical-grade stainless steel wire with incorporated prothrombic synthetic threads. The vascular occluders (Amplatzer Vascular Plug, AGA Medical, North Plymouth, MN; Canine Duct Occluder, Infiniti Medical, Haverford, PA) use nitinol mesh to create a plug or disk shape that expands within the ductus lumen to close the PDA. The duct occluders may incorporate a polyester fabric to help close the defect and promote tissue growth. Vascular plugs are placed in the lumen of the PDA, where the multiple layers of nitinol mesh result in progressive thrombosis of the vessel.[38] For these devices, intravascular access is normally established through a surgical cut-down or with the Seldinger technique. The femoral artery is usually used for arterial access, although percutaneous catheterization of the brachial artery has been reported.[39]

Use of Thrombogenic Coils

When a PDA is to be occluded using thrombogenic coils, angiography is performed before coil deployment to evaluate the shape of the ductus. Ideally, the ductus should have a distinct funnel shape (taper) to allow the coil to be lodged at the narrow end immediately before it empties into the main pulmonary artery. Thrombogenic coils should not be used in dogs with a nontapering (type III) ductus due to the risk of the coil being swept into the pulmonary vasculature.[40,41] After angiography, a guide wire is advanced through the catheter into the PDA and then the pulmonary artery. Next, another catheter housing a thrombogenic coil is advanced over the guide wire until it is in place. The thrombogenic coil is deployed under fluoroscopic observation. The coil position is evaluated, and the thorax is auscultated for changes in the nature of the heart murmur. If the cardiac murmur is still present, a second coil is deployed. This process is repeated until the characteristic murmur is no longer present on auscultation. The average patient requires two to three coils for occlusion of the PDA, but as many as 10 coils have been used.[40] For patients in which the PDA is too short (<5 mm) or fails to taper (type III), alternative techniques using another type of occluder or surgical ligation are recommended to achieve PDA occlusion.

TABLE 1 Comparison of Surgical Ligation and Thrombogenic Coil Placement for PDA Occlusion

	Coil Placement	Surgical Ligation	Advantage
Equipment costs	$500,000	$1000	Surgery
Supply inventory	$5000	$1000	Surgery
Single-use supplies	$500	$100	Surgery
Client charge (academic costs)	$2500–$3500	$2000–$2500	Neither
Procedure time	1–3 hr	1 hr	Surgery
Procedure personnel	3	2	Surgery
Animal size	Limited	Any	Surgery
PDA shape	Limited	Any	Surgery
Success rate	90%	98%	Surgery
Days in hospital	1–2	2–3 (depending on clinic)	Coils
Postoperative monitoring	Minimal	Moderate	Coils
Animal discomfort	Minimal	Significant	Coils
Mortality	<1%	<2% (experienced surgeon)–8%	Coils

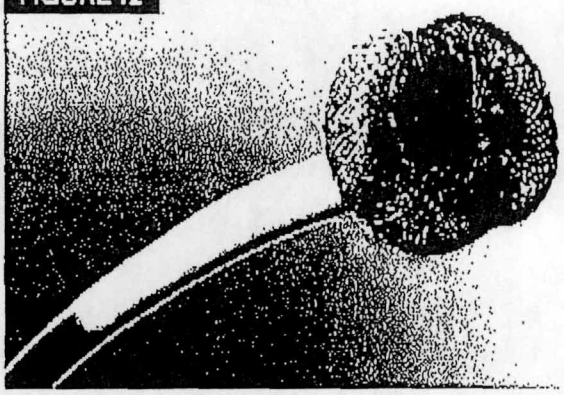
Modified with permission from Buchanan JW. Patent ductus arteriosus. In: Côté E, ed. *Veterinary Clinical Advisor: Dogs and Cats*. Philadelphia: Elsevier; 2007:820-822.

Complications reported with thrombogenic coils include coil dislodgment, inaccurate coil deployment, lameness after arterial cut-down and occlusion, significant residual flow, severe hemorrhage at the site of the arterial cut-down, pulmonary artery embolization, partial aortic deployment, hemolysis, and implant infection.[40,42] The combined rate of morbidity and mortality with coiling is less than that reported with surgical ligation (approximately 1% compared with 2% to 8%),[40] which is directly related to the surgeon's experience with PDA ligation.[4] The importance of experience should also be stressed in successful thrombogenic coil placement.

Complete PDA occlusion may be obtained in only 60% of dogs at the time of coil placement, with complete occlusion occurring in about 85% of dogs in the 90 days after placement. In most dogs with reported residual shunting, the shunting was considered "clinically insignificant."[40] However, roughly 5% of dogs receiving thrombogenic coils for PDA occlusion may require subsequent placement of additional coils to achieve complete cessation of flow.[40] Thrombogenic coils have also been used to achieve complete occlusion when there is residual blood flow after surgical ligation.[43]

A recent review of surgical ligation versus coil placement for PDA occlusion found no significant differences in procedure length or patient mortality. Surgical ligation did have a significantly higher number of "major complications" compared with thrombogenic coil placement, but the unusual nature of the complications described casts doubt on the

FIGURE 12



Courtesy of Infinio Medical

The Amplatz canine PDA device. It is made of nitinol wire mesh designed to expand distally into the pulmonary artery and proximally into the ductal ampulla.

Placement of an occluder begins with angiography to determine the size of the ductus and the pulmonary ostium. An appropriate occluder is chosen based on these measurements. A guide wire is passed through the angiography catheter and directed across the PDA into the pulmonary artery. The angiography catheter is removed, and the deployment catheter is advanced over the guide wire and across the ductus. The occluder is deployed with the distal disk opening in the pulmonary artery, and then retracted until the disk is in contact with the artery wall. Further deployment of the device fills the ostium of the PDA with the waist of the occluder. At this point, the occluder is checked for correct and secure positioning. If the operator is satisfied with the placement, the restraining cable is released and the deployment is complete. If the positioning needs to be improved, the device is reconstrained and repositioned. Correct measurement of the PDA ostium and accurate device sizing are essential for successful deployment of a duct occluding device. One review[46] reported that interventional occlusion may be best accomplished when a variety of devices are available. This report recommended the use of detachable coils for small PDAs (<4 mm in diameter) and the use of the duct occluder for larger PDAs (>5 mm in diameter).

Thoracoscopic PDA Occlusion

Thoracoscopic PDA occlusion has also been reported in dogs.[47] In five dogs, titanium vascular clips were placed to occlude the PDA. Fascia was cleared from the cranial and caudal aspects of the PDA, but dissection around the medial aspect of the PDA was not attempted. Minimal complications were encountered, and the dogs had rapid postsurgical recovery. The authors reported that the procedure was technically demanding but safe and effective. Accurate determination of the PDA diameter and vascular clip size was vital to ensure adequate occlusion. The report concluded that thoracoscopic PDA ligation was a viable alternative to surgical ligation in dogs weighing more than 7 kg with PDA diameters of less than 12 mm.[47] Other important considerations for thoracoscopic procedures include equipment investment and level of expertise with minimally invasive surgery. Thoracoscopic procedures in veterinary medicine have included persistent right aortic arch resection, lung biopsy, pericardectomy, and thoracic duct ligation. These procedures are generally reported to have decreased postoperative pain, more rapid return to function, and fewer operative site complications.[48-51] Experience with these procedures should enhance the potential for successful thoracoscopic PDA occlusion.

This aneurysm is likely due to separation of the intraaortic segment of the ductus from the lumen of the aorta by a thin flap.[3] Once the PDA is ligated, this region can still fill with blood and be visualized on radiography. If mitral regurgitation has resolved and the heart has returned to normal size, auscultation should also be normal.

Residual Flow

Failure to achieve complete occlusion and postoperative return of blood flow through a PDA are concerns in both human and veterinary medicine. In human medicine, residual flow in a PDA has potential long-term complications, including bacterial endarteritis and endocarditis of the main pulmonary artery and recanalization.[52] In humans, recanalization can occur in less than 4 months in 6% to 23% of cases involving large PDAs, the use of clips, or single or double ligation.[52,53] Secondary bacterial endocarditis has been rarely reported in dogs, but perhaps of greater concern in veterinary medicine is the potential incidence of recanalization and the return of clinical signs of PDA.[41] Recanalization has historically been cited in 2% to 3% of cases.[37] Based on return of a machinery murmur and verification at surgery, Eyster et al[37] documented the occurrence of recanalization to be 2%, with recanalization occurring twice in one dog. Lack of a murmur on auscultation does not rule out the presence of flow through a ductus vessel. One theory for recanalization is that ligation distorts the ductus, allowing the pulmonary artery and aorta to come into contact with each other. Friction from this contact could result in a new connection or fistula in an occluded ductus.[37] Recanalization most frequently occurs cranial to the ligatures, further supporting this theory.[37] Proponents of the hemoclip suggest that it does not cause distortion of the pulmonary artery–ductus–aorta orientation and thus minimizes risk of recanalization in dogs.[33] Recanalization in humans after use of a hemoclip has been documented.[33]

Another theory is that recanalization results from incomplete occlusion of the PDA.[33,53] With the increased use of color-flow Doppler imaging, residual flow may be detected in approximately 18% to 53% of cases.[17,25,31] In a human study that used an Rashkind occluder device, the largest drop in residual shunting was noted from 1 day to 6 months postocclusion due to ongoing fibrosis. This study found surgical ligation to have significantly less association with residual flow than the use of an occlusive device.[52] One author suggested that a small amount of residual flow will usually resolve by 3 months postoperatively due to the formation of scar tissue.[3] Corti et al[33] suspected spontaneous duct closure at day 560 in a case that had inadequate postoperative closure of the PDA using a hemoclip. Others disagree, stating that if residual flow is present at 1 month postligation, it is unlikely to resolve spontaneously.[41] Most agree that division and oversewing minimizes the risk of recanalization, but this technique is time-consuming and potentially increases mortality.[37] The long-term consequences of residual shunting are suspected by some to be minimal, but this topic has not been well studied in dogs to date.

Conclusion

PDA is a common condition with a variety of treatment options for occlusion. When the most appropriate option is selected and treatment is instituted early and skillfully, patients can have an excellent long-term prognosis.

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AUTHOR DISCLOSURE:

Dr. Tillson discloses that he has received financial benefits from IDEXX Laboratories and Infiniti Medical.

VetMED
EMERGENCY & SPECIALTY CARE

September 28, 2020

To Whom it May Concern:

I spoke with Mr. Evers on September 5, 2020 regarding his request for a refund following the loss of Corona. Over the course of the conversation we discussed that this procedure was performed by a highly skilled cardiology team, and despite the poor outcome the client is still responsible for the charges incurred. I pointed out that we already discounted CPR fees ($234.62), hospitalization fees ($185.97) and the necropsy and care of body ($416.21) out of courtesy. In addition, we discounted the cost of the procedure ($998.00). The owner asked if any additional discounts could be given, to which I replied no. The owner then stated that he would take VetMED and all three cardiologists to the Board if I did not agree to refund 50% of the final bill. I told the owner that I understood, but that we would not be offering any additional discounts. The owner thanked me for my time and ended the call. I have had no further interaction with the clients.

Sincerely,

Stephanie G. Foote, DVM, DACVS
Hospital Director, VetMED

Douglas A. Ducey
- Governor -

Victoria Whitmore
- Executive Director -

ARIZONA STATE VETERINARY MEDICAL EXAMINING BOARD
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INVESTIGATIVE COMMITTEE REPORT

TO: Arizona State Veterinary Medical Examining Board

FROM: PM Investigative Committee: Adam Almaraz - Chair
Amrit Rai, DVM
Cameron Dow, DVM - ***Recused***
Brian Sidaway, DVM

STAFF PRESENT: Tracy A. Riendeau, CVT – Investigations
Marc Harris, Assistant Attorney General

RE: Case: 21-29
Complainant(s): David Evers
Respondent(s): Stephanie Foote, DVM (License: 3749)

SUMMARY:
Complaint Received at Board Office: 9/16/20
Committee Discussion: 3/2/21
Board IIR: 4/21/21

APPLICABLE STATUTES AND RULES:
Laws as Amended August 2018 (Lime Green); Rules as Revised September 2013 (Yellow)

On July 21, 2020, "Corona," a 7-month-old female Maltese mix was presented to Dr. Boutet and diagnosed with a left to right shunting PDA.

On July 28, 2020, Dr. Boutet performed the dog's PDA occlusion with an ACDO device. The device was successfully deployed with no complications. While recovering after the procedure the dog went into cardiopulmonary arrest. CPR was performed but was unsuccessful.

A necropsy was performed and a definitive cause of death could not be determined.

Respondent is the responsible veterinarian for the premises.

Complainant was noticed and appeared.
Respondent was noticed and appeared telephonically. Attorney David Stoll appeared.

The Committee reviewed medical records, testimony, and other documentation as described below:

- Complainant(s) narrative: *David Evers*
- Respondent(s) narrative/medical record: Stephanie Foote, DVM

PROPOSED 'FINDINGS of FACT':

1. On July 21, 2020, the dog was presented to Dr. Boutet for a cardiopulmonary consultation f a newly noted heart murmur. The primary veterinarian identified the heart murmur during an exam for a spay procedure. The primary veterinarian wanted to get anesthetic recommendations prior to the spay procedure. Complainant reported that the dog was not exhibiting any clinical symptoms at that time.

2. Upon exam, the dog had a weight = 6.3kg, a heart rate = 130bpm, and a respiration rate = 30rpm; no temperature noted. Thoracic auscultation revealed a grade 6/6 left basilar continuous heart murmur with bounding, synchronous femoral pulses bilaterally. All other systems were evaluated and were unremarkable. An echocardiogram was performed and the findings were consistent with a patent ductus arteriosus (PDA) – the most common cardiac congenital defect in dogs. At that time, the dog's heart chamber measured normal in size and was considered a good candidate to have the PDA corrected.

3. Dr. Boutet discussed his findings with Complainant; he explained that the dog's condition resulted in an abnormal communication between the aorta and pulmonary artery and extra volume the heart sees. The dog was not currently at risk of heart failure but it was recommended to have the PDA closed as soon as possible to minimize the risk of structural changes to the dog's heart. Dr. Boutet advised Complainant that the procedure had a 98% success rate with 2% of patients having complications such as incision site infection, bleeding, and rarely device dislodgement into the lungs that would be life threatening. Any cardiac procedures have risk associated with it including anesthetic/procedural death – the risks outweigh the benefits as PDAs that are left open usually experience heart failure within a year. The procedure was scheduled for July 28, 2020.

4. On July 27, 2020, the dog was dropped off at VETMED for the PDA procedure the following day.

5. On July 28, 2020, Dr. Boutet examined the dog and found a weight = 6.3kg, a temperature = 100.3 degrees, a heart rate = 120bpm, and a respiration rate = 30rpm. Complainants reported that the dog was doing well at home with no signs of weakness, lethargy, or exercise intolerance.

6. Pre-surgical blood work was performed and was within normal limits. An IV catheter was placed and the dog was started on Plasmalyte at 31.5mL/hr. The dog was pre-medicated with midazolam and butorphanol, induced with propofol, and maintained on isoflurane and oxygen. The dog was also administered cerenia and cefazolin IV.

7. The procedure was performed by Dr. Boutet and Dr. Miller; Dr. Hubert observed the procedure and assisted with the surgical table. Dr. Matthews performed a transesophageal echocardiogram concurrently as the procedure took place. According to Dr. Hubert, the ACDO device was successfully deployed with no complications, which was confirmed via transesophageal echocardiogram and angiogram via fluoroscopy.

8. Dr. Matthews described the placement of the ACDO device; no complications occurred. After full deployment and release, appropriate positioning of the device was observed. The proximal disc was found to take its anticipated shape, but was slightly tilted in position. The finding was not uncommon in non-tapering, diminutive size ampullas. Trivial eccentric residual flow across the ACDO device was observed prior to release. After approximately one minute, no residual flow across the ACDO device was observed, and the TEE (transesophageal echocardiogram) was ended.

9. Dr. Boutet documented that the dog's murmur was resolved after the procedure. He called Complainant to advice the occlusion went well and the device was where it should be with resolution of abnormal flow. The dog was in recovery.

10. A short time later, while the dog was still intubated, the dog experienced sudden, acute respiratory arrest with production of serosanguinous foamy fluid within the endotracheal tube followed by cardiac arrest. Dr. Boutet stated the dog was moved to the CPR station where Dr. Nash took over the care of the dog. The endotracheal tube was replaced and CPR was initiated. Medications and dosages are documented in the medical record.

11. Dr. Boutet called Complainant to advise of the dog's arrest and they were trying to resuscitate her. After some time, the dog's heart restarted and responded to the atropine and epinephrine. The dog was not conscious.

12. Dr. Boutet explained that after the return of spontaneous heartbeat, thoracic radiographs and a FAST scan thoracic ultrasound were performed while the dog was hand ventilated. It was determined that the device remained in place; dislodgement was not suspected. Dr. Boutet called Complainant to discuss the radiographic findings – severe, diffuse non-cardiogenic pulmonary edema. The device was in place. He suspected severe lung reaction or an aspiration event that was not noticed or reported. After the radiographs were performed, the dog went into cardiovascular arrest again and CPR was again initiated. Complainant was advised of the dog's grave prognosis – he elected to stop manual ventilation and let the dog go. The dog passed away.

13. Dr. Boutet recommended a necropsy at Midwestern University and cardiology would cover the cost as this was not supposed to happen in PDA cases. Complainant agreed.

14. On August 14, 2020, Dr. Boutet called Complainant to report the dog's necropsy changes were consistent with CPR. The device and procedure were well evaluated with no

complication identified. He told Complainant that he wanted to speak with the pathologist regarding the areas of hemorrhage as they are unusual.

15. On August 26, 2020, Dr. Boutet called Complainant to follow up with him after speaking with the pathologist. He explained that the majority of changes were secondary to CPR. The abnormal or peculiar findings were the bleeding around one of the ocular nerves and brain stem that may represent a transient hypertensive event and hemorrhage. Alternatively, a vasculitis could not be ruled out, but would be unusual without evidence around the vessels of such a reaction although the dog may not have had enough time to develop those changes.

16. On September 5, 2020, Dr. Foote, the responsible veterinarian for the premises spoke with Complainant with respect to his request for a refund following the loss of the dog. She explained that the procedure was performed by a highly skilled cardiology team, and despite the poor outcome, Complainant was still responsible for the charges. Dr. Foote advised Complainant that they had discounted CPR fees, hospitalization fees, and the necropsy and care of the dog's remains out of courtesy. Additionally, the procedure was discounted. No other discounts would be applied.

17. Complainant expressed concerns that Dr. Boutet gave the procedure a 98% success rate. The estimate showed one injection of Iohexol; due to the ACDO not opening as normal, an additional injection of contrast was administered – this may have been an overdose of contrast. Furthermore, Complainant felt the device may not have been inserted or opened properly, leading him to believe Dr. Boutet, Dr. Miller, and Dr. Hubert did not perform the procedure properly, or know what transpired that caused the death of the dog.

COMMITTEE DISCUSSION:

The Committee discussed that it is unfortunate to lose a young pet, especially after a procedure that is typically successfully, especially when performed by experienced cardiologists. In this case, the team was very experienced and took all the proper precautions. There are reports of pets, and humans, having reactions to contrast, which is a possibility in this case. However, it will never be known since a necropsy will not pick that up. It is unknown if a patient will be allergic to contrast until you give it to them – this was the first time the dog had been administered contrast. The pathology report was very clear and descriptive, as well as the other documentation, that the device was deployed properly, thus was not the cause of the dog's death.

98% of these surgeries do great, but that means that 2% do not go well. This was Dr. Boutet's first time losing a patient as a result of this procedure. It was unfortunate that the necropsy did not show a cause of death to bring closure for all involved.

The case was handled appropriately. There only concern the Committee had was that a signature was not obtained for the authorization of surgery. Covid had likely played a part in the signature not being obtained. Some Committee members felt the surgical authorization was implied. Another Committee member commented that verbal consent can be obtained, however it needs to be witnessed and documented in the medical record; that did not occur. It would not have changed the outcome, but it is a requirement, and it would have ensured Complainant was aware of the potential risks associated with surgery.

COMMITTEE'S PROPOSED CONCLUSIONS of LAW:

The Committee concluded that possible violations of the *Veterinary Practice Act* occurred.

COMMITTEE'S RECOMMENDED DISPOSITION:

Motion: It was moved and seconded the Board find:

ARS § 32-2232 (21) as it relates to AAC R3-11-502 (H) (1) failure to obtain signed authorization from the pet owner prior to general anesthesia or surgery being performed on the dog.

Vote: The motion was approved with a vote of 3 to 0.

The information contained in this report was obtained from the case file, which includes the complaint, the respondent's response, any consulting veterinarian or witness input, and any other sources used to gather information for the investigation.

TR

Tracy A. Riendeau, CVT
Investigative Division